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THE QUEENSLAND JOURNAL OF AGRICULTURAL SCIENCE

VOL. 10. - - NO. 4.

DECEMBER, 1953.

FURTHER OBSERVATIONS ON THE USE OF THE HAMMOND SYSTEM OF PIG CARCASE APPRAISAL.

By K. J. HUTCHINSON, B.Sc.Agr. (formerly Assistant Husbandry Officer, Pig Branch, Division of Animal Industry).

SUMMARY.

Relationships between various measured carcase characters and carcase weight were established for the other Australian States as they had been previously for Queensland.

Differences in the relationships as between States are to some extent accounted for by differences in breed composition.

Examination of the grouped data from all States confirms the evidence previously given of an overall bias favouring lower weight ranges.

Arguments for and against modification of the Hammond system for Australian baconer carcase competitions are presented.

INTRODUCTION.

Following an earlier report (Hutchinson, 1951) dealing with differences between observed results and ideal measurements (Hammond system) for Queensland pig carcases, data on carcase measurements were obtained from the other Australian States and subjected to analysis.

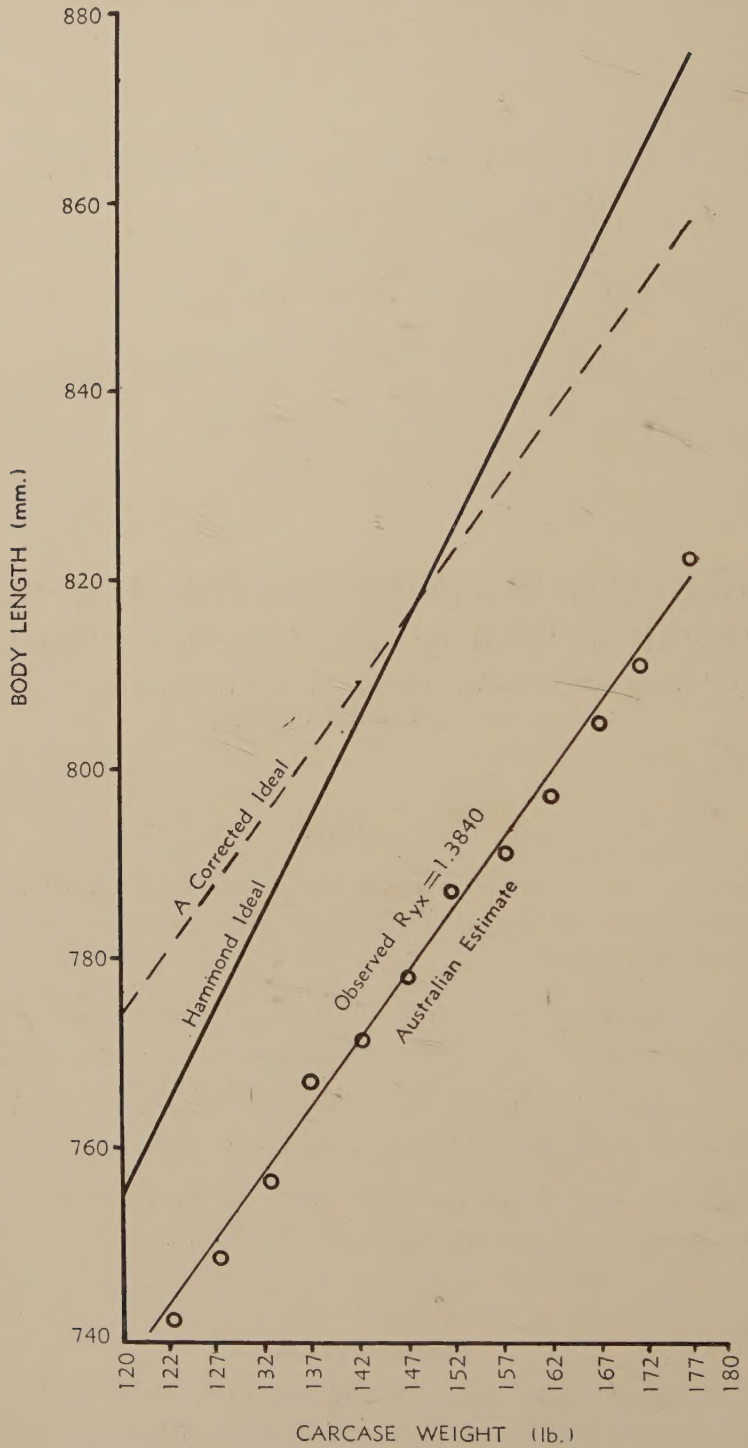

Fig. 1.

Relationship of Body Length and Carcase Weight and Suggested Correction. The adequacy of the straight-line relationship is shown by the small deviations of class means from the calculated regression line. The suggested ideal is perhaps more practical than the one suggested previously (Hutchinson, 1951).

RESULTS.

The calculated regression coefficients for the characters measured are given in Table 1.

Table 1.

CORRECTION FACTORS (R_{yx}) FOR CHARACTER VERSUS WEIGHT.

(All measurements in mm./5 lb. carcase weight).

	No. of Carcases.	Body Length.	Eye Muscle Thickness.	Backfat Thickness.	Leg Length.
Hammond Ideal	..	2·0000	0·0500	0·1000	1·000
Queensland	379	*1·3860	*0·1058	0·1428	*0·7180
New South Wales	334	‡1·3330	‡0·0900	n.a.	‡0·8750
Victoria	695	*1·4280	0·1180	*0·0579	*0·6946
Tasmania	160	‡1·2390	‡0·0521	‡0·1626	‡0·7250
South Australia	346	*1·3873	*0·0645	0·0927	0·8993
Western Australia	275	*1·3736	*0·0924	†0·0096	0·9006
Australian Estimate§	1,695	*1·3840	*0·0960	*0·0980	0·7899

* These calculated values differ significantly from the Hammond ideal (slope test).

† Regression not significant (Standard Error too large).

‡ No tests of significance available. These values were calculated by the respective State authorities. It is not known whether the regression was based on weighted means by Fisher's method.

§ Based on combined individual scores of Queensland, Victoria, Western Australia and South Australia ; individual scores of New South Wales and Tasmania were not available.

Observed and calculated figures for all the carcases included in the survey are given in Table 2, and the relationship between carcase weight and body length is shown in Fig. 1.

Differences Between States.

As outlined in the earlier report, differences in breed composition are likely to be a major factor in causing the differences between States. Evidence is accumulating on this point. For example, it is becoming increasingly evident, as results classified by breed accumulate, that the "rate of growth" for the character body length between 110 lb. and 200 lb. is highest for the Large White breed, slightly less for Berkshires and very much lower for crossbreds. This would seem to be a reflection of differences in breed development or maturity, and it is suggested as the major explanation for such results as the Victorian figure for body length (1·4280), which contained a large percentage of Large White entries, as against Tasmania (1·239) and New South Wales (1·333), whose entries contained larger numbers of crossbred and earlier maturing breeds.

Table 2.

OBSERVED AND CALCULATED MEASUREMENTS FOR 1,695 CARCASES FROM ALL AUSTRALIAN STATES (in mm.).

Weight (lb.)	Body Length.		Eye Muscle.		Back Fat.		Leg Length.	
	Obs.	Calc.	Obs.	Calc.	Obs.	Calc.	Obs.	Calc.
120–124	742·3	743·4	42·1	41·5	18·8	19·2	561·3	560·1
125–129	748·7	750·3	42·4	42·0	19·0	19·7	562·3	564·1
130–134	757·0	757·2	41·6	42·5	20·6	20·1	567·8	568·0
135–139	766·2	764·1	42·5	43·0	20·1	20·6	572·6	572·0
140–144	771·3	771·0	43·8	43·5	21·9	21·1	575·5	575·9
145–149	777·9	778·0	44·0	43·9	21·6	21·6	580·6	579·9
150–154	786·5	784·9	44·7	44·4	22·1	22·1	582·7	583·8
155–159	791·0	791·8	44·9	44·9	22·8	22·6	587·8	587·8
160–164	797·0	798·7	46·0	45·4	22·8	23·1	592·9	591·7
165–169	804·6	805·6	45·6	45·9	24·2	23·6	593·2	595·7
170–174	812·0	812·6	46·9	46·3	23·7	24·1	598·4	599·6
175–179	821·5	819·5	45·6	46·8	23·9	24·6	606·8	603·6

Summary.

Character.	Increase/5 lb.		Significant Differences.
	Ideal.	Calculated.	
Body Length	10·00	$6·9202^{+}0·1458$	Ideal greater than calculated
Eye Muscle	0·250	$0·4802^{+}0·0449$	Ideal less than calculated
Backfat	1 to 0·5	$0·4902^{+}0·0416$	No significant difference
Leg Length	5·00	$3·9497^{+}0·1733$	Ideal greater than calculated

Overall Marks.

For the Queensland analysis (Hutchinson, 1951), the important relationship between overall marks and weight range was not particularly satisfactory because of insufficient numbers, although it gave some evidence of a downward trend or lower scoring for heavy ranges. However, subsequent grouping of the results from all States has confirmed this downward trend, which provides strong evidence of an overall bias existing for measured characters, favouring lower weight ranges (Fig. 2).

This trend provides the strongest argument for change of the Hammond character weight correction table to standards which will fit observed results.

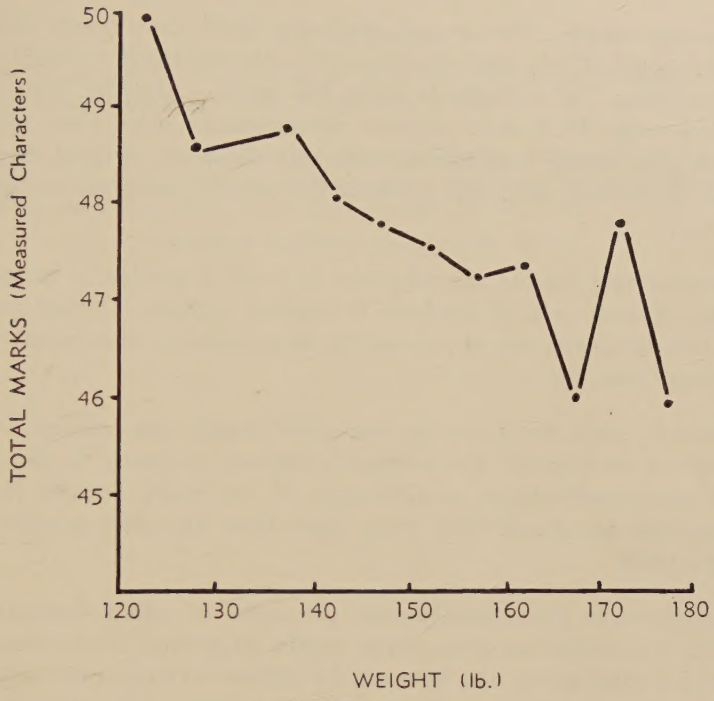

Fig. 2.

Relationship of Carcase Weight and Total Marks for Measured Characters.

POSSIBLE MODIFICATIONS OF THE HAMMOND SYSTEM.

When considering any alteration to the existing system, the fundamental principles on which the system is based must be borne in mind. If the four measured characters are considered, it may be seen that in application one of these, body length, is based on a direct measurement. Measurement of the other three characters represents an indirect method of evaluating the carcase as to the amount of muscle, fat, and bone it contains. These characters—eye muscle thickness, backfat thickness and leg length—are of use in this connection because in the statistical sense (correlation) they are related to the practical worth of a carcase.

McMeekan (1941), in detailed work subsequent to the evolution of the Hammond system, found the following correlations :—

Eye muscle depth—Weight of lean	+0·5074
Backfat thickness—Weight of fat	+0·9312
Leg length—Weight of bone	+0·7938

When the possible range of positive correlations from zero (no correlation) to +1·0000 (perfect correlation) is considered, the accuracy of these measurements as indicators can be evaluated. Such correlations are always based on large samples and are always accompanied by some variation or " scattering " in

individual measurements. For example, while the depth of eye muscle is positively related to the weight of lean, a relationship such as the one above (+0·5074) involves a large "scattering" of individuals about the mean. Actually, because of its relatively low value McMeekan suggests measurements other than eye muscle depth which give higher correlations. Backfat thickness is much more satisfactory, the correlation coefficient between this measurement and weight of fat being +0·9312.

Whatever may be the imperfections in using a particular measurement, the principle of such indirect methods is superior because (1) they are purely objective; and (2) in any case they would be more accurate than visual estimates of the weight of lean, etc.

However, when using any system based largely on indirect evaluation through various correlations, it is extremely difficult to justify in the statistical sense such fine classifications as differences of one mark between individual carcases (possible maximum 100), even apart from the error involved in the measurement itself.

In addition to these fundamental principles of carcase evaluation, any system to be of practical use over a large weight range must have some relationships by which adjustments can be made for differences in carcase weight. The most accurate correction factors which can be used are those based on the average performance of the population to which they are applied. This suitability of the Hammond correction factors to the performance of Australian competition entries is the matter in question.

Arguments For and Against Modification.

The following are submitted as some arguments for and against any modification of existing character weight correction factors as expressed in the Hammond judging tables.

For.

(1) Correction for each character which shows an individual bias should result in elimination of the overall bias illustrated in Fig. 1.

(2) Such changes do not involve any alteration in principle to the existing system, such as would be occasioned by reallocation of marks, elimination of some characters and inclusion of new, and the use of different measurements as indications of carcase value.

Against.

(1) An improvement as suggested is only a minor one, involving the general disadvantages of change in any sense. One such disadvantage would be the difficulty in making comparisons between past and future results.

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THE EFFECT OF BENZENE HEXACHLORIDE IN POULTRY FEED ON MEAT AND EGG QUALITY.

By F. N. J. MILNE, B.Sc., Assistant Husbandry Officer (Poultry), Division of Animal Industry.

SUMMARY.

A commercial form of benzene hexachloride dust (12·5% gamma isomer) and a deodorised form (99% gamma isomer) were each fed to poultry in an all-mash ration at levels equivalent to 5 p.p.m. and 25 p.p.m. of the gamma isomer to study the effect on meat and egg quality.

Groups of 20 cockerels were fed treated mashes from 12 weeks of age. The flesh was found to be tainted within 5 weeks in the group fed commercial BHC at 25 p.p.m. gamma isomer and within 7 weeks in the group fed 5 p.p.m. gamma isomer. No taint was found in groups fed deodorised BHC after 16 weeks' continuous feeding, when the experiment on flesh quality ended.

Groups of 20 pullets were fed treated mashes from 6 months of age. Taint was detected in eggs of the pullets fed mashes treated with commercial BHC after 4 weeks of feeding at the 25 p.p.m. level and after 6 weeks' feeding at the 5 p.p.m. level. In the two groups fed deodorised BHC, at 25 p.p.m. and 5 p.p.m., eggs were tainted 16 and 18 weeks respectively after feeding.

It is concluded that neither commercial BHC nor deodorised BHC should be used to control weevil infestation in grains destined for poultry feeding.

INTRODUCTION.

Excellent results have been obtained in Queensland from the use of benzene hexachloride as a protection against weevil infestation in seed maize and sorghum. In view of the likelihood of the practice being extended to grain stored for stock-feeding purposes, experiments were planned to determine the effect of feeding BHC upon the quality of eggs and flesh of poultry.

The degree of flesh taint caused by BHC has been assessed by several workers as a corollary to investigations of its use for controlling poultry ecto-parasites. Hixson and Muma (1947) showed that the flesh of poultry fed, sprayed, or kept in a house previously sprayed with crude BHC had an objectionable flavour within two weeks of treatment and that the undesirable flavour persisted for 6–10 weeks. Hannken (1950) found that, when a commercial louse powder containing 5 per cent. BHC (15 per cent. gamma isomer) was sprinkled heavily on floor litter and scratch grain was fed in this litter, a high proportion of eggs was tainted 8 weeks after the application. Absence of taint in flesh and eggs was reported by Telford (1947), who applied BHC to perches at a concentration of 1·2 per cent. gamma isomer in fuel oil, and by Milby and Lewatsch (1948), using BHC as an oil emulsion of 1·5 per cent. gamma isomer as a roost paint and as a spray for fowls.

The increasing use of BHC in the United Kingdom for the protection of grain and feeding-stuffs from insect attacks led Black, Getty, Jameson, and Pirie (1950) to investigate the risk of taints in eggs or poultry flesh when small amounts of BHC were fed continuously. A preliminary test suggested that 8 p.p.m. crude BHC (13 per cent. gamma isomer) in a laying ration would taint eggs. In a second experiment the addition of 1 p.p.m. technical grade BHC (90 per cent. gamma isomer), an amount equivalent in gamma isomer content to 8 p.p.m. crude BHC, produced 4·4 per cent. slightly tainted eggs. The results indicated that continuous feeding of 1 p.p.m. technical grade BHC was close to the threshold for producing significant taint. In neither experiment was flesh taint detected even when as much as 84 p.p.m. crude BHC was fed continuously in the ration for $6\frac{1}{2}$ months.

ADDITION OF BHC TO MASHES.

In the experiments reported here, two forms of BHC—a commercial dust ($12\frac{1}{2}$ per cent. gamma isomer) and deodorised BHC (99 per cent. gamma isomer)—were added to commercial growing and laying mashes at the rates of 5 p.p.m. and 25 p.p.m. gamma isomer. The commercial dust was added direct to the food, but bran was used as a vehicle for incorporating the deodorised BHC, an acetone solution of the insecticide being sprayed on the bran.

EXPERIMENT I.

Methods.

Five groups each consisting of 10 White Leghorn and 10 Australorp cockerels aged 12 weeks were housed intensively on new sawdust litter and were fed a commercial growing mash composed of wheatmeal, crushed oats, bran, pollard, meatmeal, buttermilk powder and salt and containing synthetic riboflavin and a fish-oil emulsion rich in vitamins A and D_3.

BHC was added to the mash as follows :—

Group 1—No BHC (Control).

Group 2—Commercial BHC equivalent to 5 p.p.m. gamma isomer.

Group 3—Commercial BHC equivalent to 25 p.p.m. gamma isomer.

Group 4—Deodorised BHC equivalent to 5 p.p.m gamma isomer.

Group 5—Deodorised BHC equivalent to 25 p.p.m. gamma isomer.

For the sake of brevity these groups will be referred to without the "gamma isomer" addendum (e.g., 5 p.p.m. gamma isomer commercial BHC will be referred to as 5 p.p.m. commercial BHC).

Five weeks after feeding commenced, and thereafter at regular intervals, two birds from each group were slaughtered. Each carcase was halved; one half was boiled and tested by a panel of three officers of the Division of Dairying accustomed to grading butter and cream, and the other half was issued for cooking under home conditions.

The method for conducting the laboratory test was as follows. The leg, breast and wing of each half-carcase were boiled until tender and then sampled by the testers, who were unaware of the treatment. A definite taint was recorded only when the individual reports of all three members agreed that tainting was evident.

Results.

A slight off-flavour was first found at 5 weeks in birds fed mashes containing 25 p.p.m. BHC (group 3), and by 11 weeks tainting was so pronounced as to make the flesh inedible (Table 1). Reports on portions of these birds roasted under home conditions indicated slight to strong earthy flavours. Tainting was reported to be most pronounced in the subcutaneous fat.

Table 1.

Results of Laboratory and Household Tests of Meat of Cockerels in Experiment I.

Treatment.	Breed.	Assessment of Taint at Stated Periods.													
		5 wk.		7 wk.		11 wk.		13 wk.		16 wk.		19 wk.		24 wk.	
		L	H	L	H	L	H	L	H	L	H	L	H	L	H
Group 1 .. (Control)	A/O	N	N	N	N	N	N	N	N	N	N	N	N	N	N
	W/L	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Group 2 .. (5 p.p.m. commercial BHC)	A/O	N	N	N	N	O	N	O	N	Treatment discontinued after 13 weeks					
	W/L	N	N	N	N	S	N	O	N						
Group 3 .. (25 p.p.m. commercial BHC)	A/O	N*	N	O	N	O	O	**	**	Treatment discontinued after 13 weeks					
	W/L	S	N	O	S	O	**	**	**						
Group 4 .. (5 p.p.m. deodorised BHC)	A/O	N	N	N	N	N*	N	N	N	N	N	N	N	N	N
	W/L	N	N	N*	'N*	N*	N	N	N	N	N	N	N	N	N
Group 5 .. (25 p.p.m. deodorised BHC)	A/O	N	N	N*	N*	N*	N	N	N	N	N	N	N	N	N
	W/L	N	N	N	N	N*	N	N	N	N	N	N	N	N	N

L = Laboratory test.
H = Household test.
N = No detectable taint.
N* = No detectable taint but musty odour.
S = Slight taint evident.
O = Objectionable taint.
A/O = Australorp.
W/L = White Leghorn.
** Not tested at home or not tested in laboratory.

Table 2.

RESIDUAL TAINT IN FLESH OF BIRDS FED FOR 13 WEEKS ON COMMERCIAL BHC.
(KEY AS FOR TABLE 1.)

Previous Treatment.	Breed.	Assessment of Taint at Stated Periods.							
		2 wk.		6 wk.		11 wk.		16 wk.	
		L	H	L	H	L	H	L	H
Group 2	A/O	N	N	N	N	N	N	**	N
(5 p.p.m. commercial BHC)	W/L	N	N	N	N	N	N	**	N
Group 3	A/O	S	N	S	N	S	N	**	S
(25 p.p.m. commercial BHC)	W/L	N	N	S	N	N	N	**	N

At the 5 p.p.m. level of commercial BHC (group 2), off-flavours were not established until 11 weeks after feeding commenced ; by 13 weeks the flesh was unpalatable. After 13 weeks, groups 2 and 3 were returned to normal feed. Three weeks later no taint was found in flesh of birds from group 2, but taint persisted in group 3 until at least 16 weeks after feeding of BHC had ceased (Table 2).

Cockerels were fed deodorised BHC mashes continuously for 24 weeks without off-flavours being recorded either by members of the tasting panel or by home consumers, although reports from both sources on a few occasions suggested " earthy " or " musty " odours.

None of the treatments had any visible effect on the health or growth of the birds.

EXPERIMENT II.

Methods.

Five groups each of 22 pullets, consisting of equal numbers of White Leghorns and Australorps which had been in production a few weeks, were used. They were fed an all-mash commercial laying ration to which the two forms of BHC were added as in Experiment I.

Eggs were tested at intervals in the laboratory by the same panel as in Experiment I. The eggs were boiled for 3 minutes and tasted without salt. Only unanimous decisions were counted. It was found that hard-boiling tended to cause masking of any flavours present. No attempt was made to grade large batches of eggs on any occasion, as it was felt that this could cause sensory fatigue to the members of the panel and so lead to inaccuracies.

In addition, all other eggs laid were distributed for normal home consumption and subsequent report on their flavour.

Results.

A report from a home source indicated the presence of taint in eggs from group 3 (25 p.p.m. commercial BHC) 4 weeks after feeding commenced (Table 4). The first panel test held 7 weeks after the experiment commenced substantiated the home report.

Table 3 sets out the percentage of tainted eggs during the 23 weeks of the experiment. Within 8 weeks nearly all eggs from group 3 (25 p.p.m. commercial BHC) were tainted. Slight taints were noted at 12 weeks in group 2 (5 p.p.m. commercial BHC) and the percentage of tainted eggs had increased rapidly by 16 weeks. It will be noted that a home consumer reported taint in eggs from group 2 six weeks after feeding commenced (Table 4). Off-flavours in eggs from pullets in group 5 (25 p.p.m. deodorised BHC) were not detected by the taste panel until after 16 weeks' feeding, but by 20 weeks at least half of these eggs

Table 3.

Results of Laboratory Tests on Eggs.

Percentage Tainted Eggs—Weeks after Treatment Commenced.			Treatment.				
			Group 1. (Control—No BHC.)	Group 2. (5 p.p.m. Commercial BHC.)	Group 3. (25 p.p.m. Commercial BHC.)	Group 4. (5 p.p.m. Deodorised BHC.)	Group 5. (25 p.p.m. Deodorised BHC.)
	7 wk.	No. Tested ..	8	10	10	10	10
		% Tainted ..	0	0	90	0	0
	8 wk.	No. Tested ..	8	4	25	3	3
		% Tainted ..	0	0	96	0	0
	12 wk.	No. Tested ..	6	7	Treatment dis-continued.	9	9
		% Tainted ..	0	14		0	0
	15 wk.	No. Tested ..	6	13		8	6
		% Tainted ..	0	76		0	0
	16 wk.	No. Tested ..	6	8		10	11
		% Tainted ..	0	75		0	36
	18 wk.	No. Tested ..	4	Treatment dis-continued.		10	17
		% Tainted ..	0			10	29
	20 wk.	No. Tested ..	4			9	11
		% Tainted ..	0			55	54
	23 wk.	No. Tested ..	4			12	11
		% Tainted ..	0			58	72

Table 4.

RESULTS OF HOUSEHOLD TESTS ON EGGS.

Treatment.	No. of Eggs Tested.	Taint First Reported—Weeks after Commencement.	No. of Eggs Reported Tainted.	Percentage Reported Tainted.
Group 1. (Control–No BHC)	1,198	..	0	0
Group 2. (5 p.p.m. commercial BHC)..	1,081	6	60	5·5
Group 3. 25 p.p.m. commercial BHC)	506	4	51	11·6
Group 4. 5 p.p.m. deodorised BHC) ..	1,023	15	15	1·4
Group 5. (25 p.p.m. deodorised BHC)	1,051	14	38	3·6

Table 5.

PERSISTENCE OF TAINT IN LABORATORY–TESTED EGGS DERIVED FROM COMMERCIAL BHC GROUPS.

Percentage Tainted Eggs—Weeks after Treatment Ceased.			Previous Treatment.	
			Group 2—5 p.p.m. Commercial BHC.	Group 3—25 p.p.m. Commercial BHC.
2 wk.	No. of Eggs		No Test	10
	% Trainted			50
4 wk.	No. of Eggs		19	10
	% Tainted		74	10
6 wk.	No. of Eggs		6	8
	% Tainted		100	0
8 wk.	No. of Eggs		7	Test Concluded
	% Tainted		57	
10 wk.	No. of Eggs		10	
	% Tainted		10	

were found to have definite off-flavours. Definite off-flavours were found in eggs from group 4 (5 p.p.m. deodorised BHC) 18 weeks after feeding commenced. The taste panel expressed the opinion that the degree of tainting in eggs from these groups was not nearly as strong as that found in eggs from group 2 (5 p.p.m. commercial BHC).

Table 4 sets out the number of eggs found to be tainted by home consumers. Although the percentages are considerably lower than those obtained from the laboratory tests, it must be borne in mind that the method of cooking could mask the presence and degree of off-flavour. It will be noted also from this table that low percentages of eggs from the deodorised BHC groups were reported as being tainted by home users.

Table 5 shows the frequency of taints in eggs laid by groups 2 and 3 at intervals after the feeding of commercial BHC ceased. Some tainting was still detected in laboratory tests at the close of the experiment, 10 weeks after the feeding of 25 p.p.m. commercial BHC had ceased.

The egg production of each group was recorded for the period of the test, no marked differences in production between groups being found. The health of the birds did not appear to be adversely affected by the various treatments of commercial or deodorised BHC.

DISCUSSION.

The results obtained from the experiments show that commercial BHC when added to either growing or laying mashes at a rate equivalent to 5 p.p.m. or 25 p.p.m. gamma isomer produced definite taints in both eggs and poultry meat.

Black *et al.* (1950) reported absence of taint in flesh of birds fed for $6\frac{1}{2}$ months on a ration containing 84 p.p.m. crude BHC, which approximately equalled the lower level of commercial BHC (5 p.p.m. gamma isomer) used in the experiments reported here. Methods of cooking were similar and the portions tasted were identical. The results obtained through the technique adopted in conducting these tests in the laboratory leave no doubt that the use of commercial BHC at a level equivalent to 5 p.p.m. gamma isomer in poultry feed produces strong and objectionable taints.

Not all cockerels in group 3 (25 p.p.m. gamma isomer) were adversely commented on by home users, although portions from the same carcases tasted under laboratory conditions were found to be objectionable and unpalatable after 7 weeks of feeding (Table 1). It is possible that the method of home cooking, in most cases roasting, tended to mask the presence of taint.

When tainting was definitely established in flesh and eggs from groups fed either level of commercial BHC, treatment was discontinued, and it would appear that the degree of residual tainting and persistence over a period of time is related to the amount of commercial BHC ingested over the test period.

With regard to deodorised BHC (99 per cent. gamma isomer), taints were found in eggs but not in poultry meat, although laboratory and home reports on carcases from groups ingesting the two levels of this form of BHC suggest occasional " earthy " or " musty " odours.

Black *et al.* (1950), using a technical grade of BHC (90 per cent. gamma isomer), also recorded some degree of tainting in eggs when used at a much lower level than the 5 p.p.m. gamma isomer deodorised BHC in this work.

In both experiments and at both levels of commercial and deodorised BHC, laboratory findings on eggs were confirmed by home users' reports.

From these experiments it is concluded that the objectionable flavours encountered in eggs and flesh and resulting from the use of commercial BHC at the rates of 5 p.p.m. and 25 p.p.m. gamma isomer respectively in mashes, and definite taints in eggs associated with the use of deodorised BHC at the same rates, render these preparations totally unsuited for use as insecticides in grain destined for poultry feeding.

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PEANUT PRE-EMERGENCE AND CROWN ROT INVESTIGATIONS.

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SUMMARY.

The results of investigations covering a number of years are reported.

Crown rot is caused by Aspergillus niger. Pre-emergence rot is due to Rhizopus arrhizus, Aspergillus niger and probably other organisms.

Seed treatment, particularly with organic mercurial fungicides and the organic fungicide thiram, improves field emergence, sometimes to a remarkable extent.

The previous cropping history of the soil has a marked influence on losses from pre-emergence and crown rots.

INTRODUCTION.

Shortly after the development of an extensive peanut industry in the South Burnett district of Queensland, growers met with serious difficulties in establishing good stands of this crop (Fig. 1). Losses occurred in several ways. Numbers of the kernels merely swelled and rotted in the soil. Evidence has been obtained that many of these were viable. Other kernels germinated but failed to emerge, having rotted below ground level. Still more emerged but failed to survive to maturity due to the development of the disease now known as crown rot. It was these losses which led to the investigations reported in this paper.

SYMPTOMS AND ASSOCIATED ORGANISMS.

Crown rot may affect peanut plants at any stage from the seedling to the mature plant, but is most common in the former. The first obvious symptom is a wilt of the whole plant, or in the case of large individuals a wilting of one or more branches. The stalk and root tissues of affected plants, just below the ground, are dark and shrunken and have a somewhat shredded appearance. Black masses of spores of *Aspergillus niger* Van Tieghem can usually be readily seen on the surface of the affected area. Most wilted plants soon die but some maintain a precarious existence on adventitious roots sent out from the stem above the affected area.

Fig. 1.

A Peanut Crop in which the Stand has been Thinned Out by Crown Rot.

Associated with crown rot there is a pre-emergence rot in which rotting of the seed or hypocotyl kills the plant before it appears above the ground. This is often due to the same fungus that causes crown rot, but frequently a type of pre-emergence rot with somewhat different symptoms is found. This is distinguished by the presence on the rotting seed of a loose mat of mycelium in which is incorporated a mass of soil particles. The mycelium is that of a species of *Rhizopus* corresponding closely to *R. arrhizus* Fischer, as described by Hildebrand and Koch (1943). (G. D. Bowen, Queensland Department of Agriculture and Stock unpublished records, 1953). It is also found occasionally on the rotted hypocotyl of plants which have the aboveground symptoms of crown rot.

Observations and inoculations reported later indicate that in the disease complex plants showing crown rot symptoms are usually infected with *A. niger* with an occasional *R. arrhizus* infection. In pre-emergence rot, either organism can be involved; the evidence suggests that *Rhizopus* occurs more frequently in this phase of the disease. In a few instances other organisms may be involved.

PATHOGENICITY TRIALS.

Isolations from affected plants have yielded a variety of organisms. Unidentified species of bacteria are common, while the most frequently occurring fungus is the one responsible for the black spore masses, namely *A. niger*. *R. arrhizus* and *Pencillium* spp. appear on numerous occasions and *Botrytis* sp. and *Fusarium* sp. occasionally.

Whole nuts were selected for freedom from injury and surface sterilized by soaking for 30 minutes in 1–400 commercial formalin. They were then hand-shelled carefully to avoid contamination. Before planting the seed in autoclaved soil, the appropriate inoculum was applied either as a fragment of an agar culture or as a water suspension of spores. Some of the kernels were first injured with a small cut or abrasion.

Attempts to reproduce the disease with various unidentified bacteria were all negative. An occasional lesion was found after inoculation with *Penicillium* sp. but not consistently enough to warrant any conclusions on the pathogenicity of this organism.

Very few peanut kernels germinated after inoculation with *R. arrhizus*, the seed usually rotting without any growth (Fig. 2, B and C). It developed the mat of mycelium and adhering soil characteristic of this type of pre-emergence rot. The fungus was re-isolated from affected seed. Under moist conditions in the plant house, *R. arrhizus* spreads rapidly through and over the surface of the soil. In one series of inoculations with various organisms, in which tins of soil were used and placed close together in an incubator, the spread of this fungus to adjacent tins spoiled the trial. No sudden wilt of established plants has been obtained by inoculation of seed with *R. arrhizus*.

Typical symptoms of crown rot were produced only when *A. niger* was used as inoculum (Figs. 2A, and 3). The organism was readily re-isolated from affected plants. However, the disease could not be induced at will. Only a small proportion of the seed inoculated developed into diseased plants, the majority producing normal plants. Attempts to increase the amount of disease by varying the soil moisture content were not successful. Field observations indicate that soil texture influences the development of the disease, but this aspect has not been fully explored in the laboratory.

Figures from inoculation experiments are given in Table 1.

The isolates of *R. arrhizus* used consistently reduced emergence and few plants survived after infection. These few did not subsequently develop crown rot. Emergence was variable after infection with *A. niger*, and usually a few

A B

C D

Fig. 2.

Artificial Inoculation of Peanut Kernels with the Organisms Responsible for Pre-emergence Rot and Crown Rot. A, *Aspergillus niger*. B, *Rhizopus arrhizus* ex Peanut. C, *R. arrhizus* ex Apricot. D, uninoculated.

Fig. 3.

Crown Rot Resulting from Artificial Inoculation of Peanut Kernels with *Aspergillus niger*.

Table 1.

EMERGENCE OF PEANUT KERNELS INOCULATED WITH VARIOUS ORGANISMS. (MEAN EMERGENCE PER 10 PLANTS.)

Organism.	Experiment A.		Experiment B.	
	Injured.	Uninjured.	Injured.	Uninjured.
Aspergillus niger	1·1	0·5	7·8	7·4
Rhizopus arrhizus from peanut ..	0·3	0·0	1·0	2·0
Rhizopus arrhizus from apricot fruit ..	0·1	0·1	..	..
Penicillium sp.	..	..	7·8	7·8
Unidentified bacteria	..	..	6·2	7·6
Not inoculated	4·0	4·0	6·8	7·2

of the surviving plants wilted and died with the development of stem lesions in the cotyledonary region. These carried the spores of *A. niger*. The evidence suggests that *Rhizopus arrhizus* is responsible for most of the pre-emergence rot and that *Aspergillus niger* is the cause of crown rot.

KERNEL INJURY.

A suggestion was prevalent at one time that injury to the kernels was directly responsible for the failure to emerge and loss of seedlings. Self-sown peanuts are generally not affected with crown rot nor are plantings of whole nuts. Exceptionally the disease may be found in such plants but the amount is usually small. Furthermore, carefully hand-shelled and hand-planted kernels develop little if any crown rot. Figures comparing hand-shelled with machine-shelled kernels taken from an experiment described later are given in Table 2.

Table 2.

PERCENTAGE EMERGENCE OF HAND-SHELLED AND MACHINE SHELLED KERNELS.

Treatment.	Trial A.	Trial B.
Hand-shelled	70	76
Machine-shelled	44	49

It is evident that injury to kernels by the shelling machinery has a considerable bearing on the disease, although this has not always been verified when deliberate injuries were induced in the laboratory.

Investigation of planting machinery indicated that it did in fact visibly damage a number of kernels. However, it has been found that machine-shelled and hand-planted kernels are affected to an extent comparable with that of machine-shelled and machine-planted kernels. This indicates that the injury occurring in the sheller predisposes the kernel to the troubles under consideration but that injury in the planter has little if any further effect.

Consideration of the injury factor leads to several solutions of the crown rot problem but considerations of cost and other difficulties have precluded their adoption. Whole nuts could be used for seed. However, the distribution of plants in the row is poorer with whole nuts and they require more moisture for germination. This can only be partly overcome by pre-soaking the nuts. The objection to hand-shelling is the labour involved in the operation.

SEED TREATMENT.

It was considered that machine-shelled kernels would germinate more satisfactorily if chemically treated, and trials to determine a suitable treatment were started. Early trials consisted of laboratory plots using small quantities of seed. It was found that applications of formalin for 12 hours at strengths of 1 part of formaldehyde to 4,000 parts of water and stronger seriously reduced germination of peanut kernels. A 10-minute dip with a strength of 1 part to 500 parts of water or weaker was without deleterious effect, and the shorter dipping period was adopted for further trial. Bluestone and corrosive sublimate were tried at various strengths and the 1 per cent. and 0·1 per cent. dilutions respectively were adopted as the rates most likely to be useful. Mercury and copper dusts were most promising, particularly organic mercury dust.

The seed treatments were then field-tested using single drills $2\frac{1}{2}$ chains long with eight replications. Except where otherwise indicated, the plots were planted with a double-row planter, using weighed amounts of seed. By weighing the excess seeds from each row and counting the kernels per pound, the number of seeds per row was estimated. The stands were counted and results are reported in Table 3 as percentage emergence. Figures for the laboratory germination are also given.

Table 3.

SEED TREATMENT TRIAL, No. 1.

Treatment.	Laboratory Germination.	Field Emergence.
	%	%
Mercurial dust A	96	81
Mercurial dust B	93	74
Corrosive sublimate—		
1 : 1000 for 3 minutes ..	87	68
Bluestone—		
1 : 100 for 3 minutes	72	60
Formalin—		
1 : 240 for 10 minutes ..	55	44
Copper carbonate dust	83	68
Untreated	74	48
Untreated hand-planted	..	51
Necessary differences for significance:		
5% level	..	6·2
1% level	..	8·3

The two mercurial dusts were experimental fungicides containing organically combined mercury in undisclosed proportions. The dusts were applied at the rate of 1 oz. to 20 lb. of seed. For the corrosive sublimate treatment, kernels were dipped in a solution of 1 part per 1,000 for three minutes, then rinsed in water. For the bluestone dip the rinsing was omitted. After dipping, the seed was dried and stored till required.

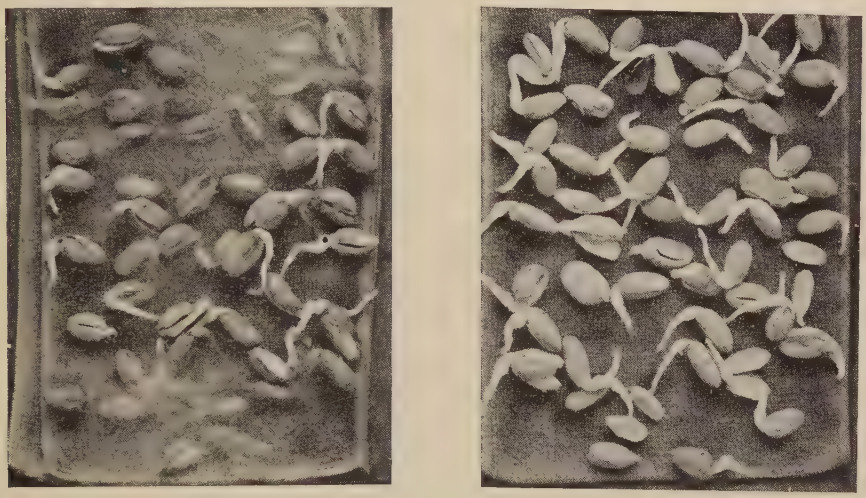

Fig. 4.
Effect of Mercurial Dusts on the Germination of Peanut Kernels. Untreated seed on left.

The mercury dusts were outstanding and this type of dust has since proved most consistent in improving field emergence.

The corrosive sublimate and bluestone dips resulted in delayed germination and the formalin in seriously delayed germination. On account of this effect and their inferiority to the organic mercury materials, they were not again tested. The copper dust was somewhat inferior to the mercurials but was considered to warrant further testing.

A further field trial of treated peanuts consisted of short drills in which 25 kernels were planted per drill and subsequent emergence counted. Four treatments were tested against untreated in four randomised blocks. The results obtained are shown in Table 4.

The two organic mercury dusts were again superior to the copper dusts and the latter were not given further consideration.

Following this work, seed treatment with mercury dusts was adopted as a standard practice in the industry in 1937, with marked reduction in disease incidence. The treatment is applied in bulk by the Queensland Peanut Marketing Board, which handles practically all the peanut seed in the State.

Table 4.

SEED TREATMENT TRIAL, No. 2.

Treatment.	Field Emergence.
	%
Ceresan (1·5% Hg)	67
Agrosan (1·5% Hg)	65
Cuprocide	52
Copper carbonate	41
Untreated	33
Necessary differences for significance :	
5% level	11·5
1% level	16·8

Seed Treatment with New Organic Fungicides.

When new organic fungicides became available, two of them were compared with the standard treatment, and a mercury dust containing only 0·5 per cent. Hg was included. The results from 12 replications in single drills with 100 seeds per drill were as shown in Table 5.

Table 5.

SEED TREATMENT TRIAL, No. 3.

Treatment.	Field Emergence.
	%
Ceresan (1·5% Hg)	72
Ferbam (80% dust)	73
Mercury dust (0·5% Hg)	59
Thiram (50% dust)	75
Untreated	60

The dust with the lower percentage of mercury was unsatisfactory but the organic preparations were among the best. Arrangements were made for a further trial of thiram, which was considered to have the most desirable properties. In these trials some new materials were incorporated and various amounts of dusts were tested on three different kinds of soil. The same seed was used throughout. The results are given in Table 6. A discussion of the effect of different types of soil will be found in a later section.

Table 6.

PERCENTAGE FIELD EMERGENCE IN A RATE-OF-TREATMENT AND ROTATION TRIAL.

Previous Crops.	Rhodes Grass.			Peanuts with Cover Crops.			Peanuts without Cover Crops.			Mean.
Oz. per 100 Lb. Kernels.	10.	5.	2·5.	10.	5.	2·5.	10.	5.	2·5.	
Ceresan	68	76	76	66†	62†	55	78†	69†	58†	68
Agrosan	76	73	77	71†	67†	65†	71†	73†	66†	71
Thiram	72	74	77	67†	64†	60†	51	54*	49	63
Hexachlorbenzene	76	67	62	49	43	48	39	38	35	49
Untreated	..	71	..	..	47	..	..	38	..	49

* Significantly better than untreated at 5% level.

† Significantly better than untreated at 1% level.

Ceresan and Agrosan, the two organic mercury dusts, again proved superior to the other fungicides. Hexachlorbenzene, which is a highly efficient preventive of wheat bunt, was useless for peanuts. The performance of thiram varied somewhat, and as this material was at the time much more expensive than the mercurials, its commercial use could not be recommended. There appeared to be rather less post-emergence loss of plants after treatment with thiram than after treatment with Ceresan or Agrosan.

The use of 10 oz. of dust per 100 lb. of seed did not result in any improvement over the standard rate of 5 oz. per 100 lb. With half this rate (2½ oz. per 100 lb.) there was still a high degree of disease control but not as high as with the standard rate.

A 5 x 4 randomised block trial was laid down to test two brands of chloranil (tetrachloroparabenzoquinone). Each plot comprised five rows of 50 kernels each. The results as percentage field emergence are given in Table 7.

Table 7.

SEED TREATMENT TRIAL, No. 4.

Treatment.	Field Emergence.
	%
Ceresan	81
Agrosan	77
Tetroc (chloranil 98%)	72
Spergon (chloranil 98%)	68
Untreated	60
Necessary differences for significance :	
5% level	7·2
1% level	10·3

The two brands of chloranil (Tetroc and Spergon) both had some beneficial effect but neither was as good as the organic mercurial preparations. Chloranil was therefore not considered further.

It is interesting to note the rather high figures for field emergence of the untreated plot in this trial. This is accounted for by the use of good seed in a paddock newly ploughed after being for several years under Rhodes grass. The trials with seed lots of varying germinability reported in Table 11 were planted at the same time and place. Even the poorest of these seed lines showed a 60 per cent. emergence.

Four of the replications in the first treatment trial were planted with seed which had been recently shelled, the other four with kernels which had been shelled from two to four weeks before treatment and sowing. There were indications that the delay in planting after shelling somewhat reduced the emergence of untreated seed, but this effect, if real, was nullified by the best treatments.

Figures for selected treatments are given in Table 8.

Table 8.

Effect of Time of Shelling on Field Emergence.

Treatment.	"New" Seed.	"Old" Seed.
	%	%
Mercurial A	79	82
Mercurial B	75	73
Untreated	55	44
Untreated, hand-planted	57	38

Effect of Seed Treatment on Yield.

Any increase of yield from seed treatment may be due to improved stand or to increased average yield per plant. In an experiment planned to obtain information on these points untreated and Ceresan-treated seed was planted at from 22 lb. to 56 lb. of kernels per acre. Two randomised blocks using six seeding rates and split plots for the treatment were planted. The sub-plots were six rows wide and ran right across a paddock in order to facilitate planting. A 10-chain length was marked off for harvesting and threshing and weights were taken for the total yield of nuts from the 10 chain x 18 feet sub-plots. Stand counts at various intervals and other data were also recorded and are summarised in Table 9.

The treatment had a marked beneficial effect on yield. This appeared to be only partly due to increased stand, and in fact there was no statistically significant increase in yield with increasing planting rate. Under the conditions of this experiment a stand of one plant per foot of row could be achieved by planting about 30 lb. of treated seed per acre. It could with difficulty be approached by planting 56 lb. of untreated seed. Seed treatment is considerably less costly than increasing rate of seeding and results in a more uniform stand ; this could account in part for the higher yields from treated seed.

Table 9.

SEED TREATMENT AND YIELD.

Planting Rate.	Stand (Plants per Chain Row).		Yield (Lb. per Acre).	
	Ceresan.	No Dust.	Ceresan.	No Dust.
22 lb. per acre	42	25	1,260	970
26 „ „ „	54	31	1,150	1,060
30 „ „ „	69	40	1,310	1,010
36 „ „ „	71	39	1,340	1,170
44 „ „ „	79	50	1,240	1,100
56 „ „ „	102	55	1,120	1,090
Mean	69	40	1,230	1,070
Necessary differences for significance :—				
Between planting rate means with same sub-plot treatment :				
5% level	..	..	230	230
1% level	..	..	350	350
Between marginal treatment means :				
5% level	2·8		86	
1% level	4·3		130	

Seed Treatment and Germination Tests.

The growth of *Rhizopus* sp. and *Aspergillus niger* on peanut kernels seriously interferes with the laboratory determination of percentage germination. *Rhizopus* sp. in particular tends to spread on the germination tray and affect considerably more kernels than those originally infected. Because of this it is difficult to obtain reproducible results, but the difficulty is overcome by treating the seed with an organic mercury dust. Table 10 illustrates the variation in tests of two samples of the same seed lots..

Table 10.

VARIABLE PERCENTAGE GERMINATION IN LABORATORY TESTING.

Untreated.		Treated.	
1st Test.	2nd Test.	1st Test.	2nd Test.
39	92	98	95
58	90	97	97
57	87	97	96
6	89	96	97

In practice, peanut kernels intended for seed are after shelling submitted for germination test and those seed lots which fail to reach the required standard of 80 per cent. after treatment are discarded. However, emergence in the field varies with many other factors besides laboratory germination of the seed. When planted in unsuitable soil, treated seed giving a good laboratory germination may still show a poor field emergence. When planted in soil of a good type this difference is less marked, as can be seen from Table 11. Under these circumstances laboratory germination of treated seed gives a satisfactory prediction of field performance.

Table 11.

LABORATORY GERMINATION COMPARED WITH FIELD EMERGENCE IN A SCRUB SOIL OF GOOD TYPE.

Untreated.		Treated.	
Germination (%).	Emergence (per 100 Seeds).	Germination (%).	Emergence (per 100 Seeds).
73	34	94	68
74	38	94	78
61	36	93	70
78	28	96	62
71	52	91	84
76	32	95	64
76	52	97	80
Mean 73	39	94	72

It is obvious that, while conditions in the field modify the laboratory performance of seed, it is still very important to have seed of a high standard of germination. It is not possible to test this standard with reasonable accuracy unless the seed is first treated with an efficient fungicide.

CROP ROTATION AND CROWN ROT.

Reference has been made to the influence of soil type and crop rotation on crown rot and associated diseases of peanuts. This influence is very marked. Direct evidence on this point was obtained and is shown in the figures of Table 6.

On a good scrub soil newly ploughed after several years under Rhodes grass, there was little benefit from seed treatment. On a scrub soil cropped to peanuts each year and on which a winter crop was ploughed in between peanut crops, the overall stand was poorer and differences due to treatment were marked. On a forest soil under continuous peanut culture, the differences were greater still.

Numerous observations support the claim that crown rot is more prevalent when the organic matter of the soil has been depleted. The high incidence of disease in old peanut lands could be explained therefore on the basis of either the depletion of organic matter or a build-up of pathogens in such land. However, in many such paddocks good stands of peanuts are frequently to be found only on areas where peanuts have been stacked and threshed in previous seasons. These areas should carry larger rather than smaller concentrations of pathogens, and in fact wilt diseases occur more frequently in them than elsewhere. Pre-emergence rotting and crown rotting are conspicuously less prevalent in soil containing the peanut debris from the old stacks. The evidence all points to a lower incidence of these diseases when humus is abundant, however it is derived.

DISCUSSION.

Crown rot of the peanut due to *Aspergillus niger* does not appear to have assumed the serious proportions in other parts of the world that it has in Queensland. Jochems (1926) recorded the fungus in peanuts in the East Indies and reported positive results on inoculation but dismissed the disease as unimportant. Gibson (1949) reported the disease as locally severe in East Africa and has since (1950) obtained successful inoculations with *A. niger*. He also recorded *Rhizopus arrhizus* among the fungi found in typical diseased material. Crosier (1944) recorded *Rhizopus nigricans* as severely injuring peanut seedlings in the germinator.

The incidence of crown rot in the South Burnett district in Queensland can be such as to constitute a serious threat to the peanut industry there, while more recently the disease has appeared to a serious extent in some North Queensland peanut crops. However, with adequate precautions of seed treatment and crop rotation as outlined previously (Morwood, 1946) the disease may be kept down to small proportions.

In the field there is inevitable confusion between crown rot and pre-emergence rot caused by *R. arrhizus*. The two diseases combine to reduce stands and the benefit of seed treatment is attained by a degree of control of both troubles.

Seed treatment of peanuts has been widely adopted for the improvement of field emergence. Gibson (1950) in East Africa and Prevot and Commun (1951) in French Equatorial Africa found that seed treatment increased establishment by up to 50 per cent. and the latter workers stated that seed disinfection is one of the most effective means of developing ground nut production. Hopkins (1945) refers to additional control of root rot caused by *Sclerotium rolfsii* by seed disinfection with mercurial dusts. This effect has not been observed in Queensland.

Reference to control measures for crown rot and pre-emergence rot has already been made and it is now only necessary to reiterate the salient points. Machine-shelled kernels should be treated before planting with an organic mercury dust with a minimum mercury content of 1·5 per cent. at the rate of 5 oz. per 100 lb. kernels. With Red Spanish kernels the rate should be reduced to 2½ oz.

per 100 lb. Peanuts should be sown in a rotation that ensures ample organic matter in the soil. This is most likely to be attained by including several years of Rhodes grass pasture in the rotation. Peanuts should be the first crop sown following the ploughing out of the Rhodes grass. Crop residues should be incorporated in the soil and not burnt (Kerr and Cartmill, 1951).

ACKNOWLEDGMENT.

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NOTES ON TWO PINEAPPLE DISEASES IN QUEENSLAND.

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SUMMARY.

The isolation of Thielaviopsis paradoxa (De Seynes) von Höhn from white leaf spot lesions and the reproduction of typical symptoms by inoculation with this fungus are described.

The results of inoculations with a Fusarium and two types of Penicillium isolated from fruitlet core rot are given. Circumstantial evidence is presented that mealy bugs and mites provide a means of entry of the causal organism.

WHITE LEAF SPOT.

White leaf spot, or " white leaf," as it is often known, is a common disease of pineapples in Queensland but one which rarely causes serious damage.

Its occurrence is usually restricted to the months of March to May and then only to periods of overcast, rainy weather. It is most prevalent in young, quick-growing plantations, rarely attacking the harder foliage of older or ill-nourished plants.

Symptoms.

The first symptom is a small yellow to brown spot on the leaf which rapidly elongates under moist conditions. During prolonged periods of rainy weather, the lesions may reach several inches in length and spread right to the tip of the leaf. If the lesion spreads across the leaf and girdles it, the part above droops over and withers (Fig. 1).

The extent of the damage depends on the duration of the favourable conditions, since fine weather results in a rapid drying of the affected areas to give straw-coloured, or almost white, papery lesions. The margins of the spots often remain brown in colour and discoloured areas may extend through the lesions.

On inspection, the lesions will be seen to commence where two leaves have rubbed, or where a leaf has broken over, been punctured by an insect, or been injured in some other manner.

Fig. 1.

Pineapple Leaves Showing Natural Infection with White Leaf Spot.

Causal Organism.

According to Cook (1933), white leaf spot was reported by Fawcett from Puerto Rico in 1908, and Nowell (1922) also described the symptoms. In Hawaii, Larsen (1910) attributed the disease to a wound infection by *Thielaviopsis paradoxa* (De Seynes) von Höhn ; he published experimental work showing the manner of infection and describing the associated weather conditions. However, Cook (1933) could not isolate the causal fungus in the Puerto Rico area, and suspected weather conditions as being responsible, without the influence of any other agency.

Although white leaf spot was described in Queensland by Lewcock (1947), the causal organism was not identified.

In the autumn of 1952 a series of isolations was made from the margins of white leaf spot lesions by the author, and in a number of cases *Thielaviopsis paradoxa* was obtained amongst the isolates.

Only small developing lesions were selected for isolation work, which was timed to coincide with periods of suitable weather for leaf spot development. It was found that with the onset of fine weather the organism rapidly died out in the leaf tissue and isolation attempted then was fruitless.

Inoculation of healthy leaves on potted plants was carried out in the following manner.

The leaves were surface sterilized with corrosive sublimate and 12 inoculations were made by pricking with a sterile needle through a spore suspension of *T. paradoxa* in a drop of sterile water. In another 12 cases a drop of spore suspension was placed on the leaves without subsequent injury. The controls consisted of plants on which 12 leaves had been pricked through drops of sterile water. All plants were placed under bell jars.

In 48 hours, oblong, light-brown lesions with dark-brown margins had developed at each point where inoculation included injury, and in five days these lesions were several inches long. No lesions developed where the leaves were not pricked or in the controls (Fig. 2).

Fig. 2.

Result of Inoculating Pineapple Plants with *Thielaviopsis paradoxa*. *Left*—Four leaves pricked without inoculation. *Right*—Three leaves pricked and inoculated.

Isolations were made from the margin of each lesion and *T. paradoxa* obtained in every case.

Once the bell jars were removed from the inoculated plants, the affected areas rapidly dried out and assumed the pale, papery appearance which is typical of the disease in the field.

Re-inoculations were made into healthy plants using the same technique as before, but as the work was timed to coincide with showery weather, one treated plant was not placed under a bell jar. Both the plants inside and outside the bell jars, however, developed lesions and re-isolation of *T. paradoxa* was again successful.

Discussion.

Thielaviopsis paradoxa may be commonly found in pineapple plantations during the summer months growing on decaying fruit, tops and other pineapple material, and thus there is an ample supply of inoculum for infection. As the fungus, however, is purely a wound parasite of pineapples and requires special weather conditions before attacking the injured leaves, field damage is not serious. Control measures would, therefore, be rarely if ever required and experimental work in this direction has not been considered necessary.

FRUITLET CORE ROT.

Fruitlet core rot, also commonly known as " brown spot " or " brown rot," was the first fungous disease of pineapple fruit to be recorded in Queensland. It was first investigated by Tryon in 1898. Since then the disease has appeared sporadically in all pineapple-growing districts in the south of the State.

The disease affects fruit of all varieties of pineapple but is most severe in the Ripley Queen. Its seasonal incidence is variable, although it is usually most prevalent in fruit maturing in the winter or spring.

Symptoms.

Fruitlet core rot is often not detected until the fruit is cut, but the failure of one or more fruitlets to colour with the rest at maturity is usually an indication that the disease is present. Some badly affected " eyes " become brown and sunken as the fruit ripens, and brown fissures may appear between the fruitlets. This is particularly the case if the fruit is held until it is over-ripe.

Internal symptoms consist of a browning of the centre of the fruitlet starting immediately below the floral cavity, and in severe cases extending to the core. The lesions are firm and vary in size from a small speck to a rotten area involving the whole of one or more fruitlets.

Fig. 3.

Fruitlet Core Rot. Natural infection.

Causal Organisms.

Tryon, following his investigations in Queensland (Tryon, 1898), attributed fruitlet core rot in the Ripley Queen variety to injuries caused by a Tarsonemus mite followed by infection with *Monilia* sp. The disease in the Smooth Cayenne variety appeared to differ in that the associated organism was a *Penicillium*. Mealy bugs, although present occasionally, were not thought to be implicated. Tryon considered later (Tryon, 1928) that minute growth cracks also provide the necessary injury in smooth-leaf pines. Later, Simmonds (Veitch and Simmonds, 1929) associated a *Penicillium* and a *Fusarium* with the disease in the Ripley variety.

Larsen in Hawaii (Larsen, 1910) found a *Fusarium* to be the chief causal organism there, and Linford (1952) reported fungi of the genera *Penicillium* and *Fusarium*, as well as certain yeasts and bacteria, to be capable of causing the disease in Mexico.

Recent Investigations.

During 1951–1952, growers in the Brackenridge district near Brisbane experienced considerable losses of Ripley Queen pineapple fruit from fruitlet core rot. The disease, although present all the year round, appeared to be most prevalent in the winter fruit, and was worse in ratoon than in plant crops.

Mealy bug (*Pseudococcus brevipes* (Cockerell)) infestation was evident on both the plants and the fruit in the affected plantations. Few fruit did not at least have the insects present on the attached leaves. The infestation had increased during the 1951 drought and populations were greater in the ratoon than in the plant crops.

Four longitudinal sections were made through each fruit examined. It was found that many of the floral cavities under the "eyes" contained mealy bugs. Counts were made of the eyes affected by fruitlet core rot and of the eyes containing mealy bugs. These figures are recorded in Table 1. It is seen from this table that 96·3 per cent. of the eyes affected by fruitlet core rot contained mealy bugs, while only 69·2 per cent. of the total number of eyes examined were infested. There appears to be some association between mealy bug infestation and the presence of fruitlet core rot.

Table 1.

Association of Mealy Bugs and Fruitlet Core Rot in Pineapple Fruit.

Serial No. of Fruit.	Number of Fruitlets Examined.	Number Containing Mealy Bugs.	Number with Fruitlet Core Rot.	Number of Affected Fruitlets Containing Mealy Bugs.
1	19	14	5	5
2	23	17	9	9
3	22	16	1	1
4	19	18	14	13
5	27	15	8	8
6	25	14	11	11
7	21	11	3	2
8	20	19	8	8
9	17	14	4	4
10	24	20	4	4
11	19	13	1	1
12	25	2	2	1
13	18	17	2	2
14	24	21	5	5
15	18	11	3	3
Totals— 15	321	222	80	77

Percentage of fruitlets containing mealy bugs .. .. 69·2%

Percentage of diseased fruitlets containing mealy bugs .. .. 96·3%

Investigation of the Causal Organism.

Isolations were made from infected fruitlets and a *Fusarium* and two types of *Penicillium*, differing in cultural characteristics, were obtained.

Fruit nearing maturity were then surface-sterilized and inoculated on the plant with each of the fungi isolated. Each eye to be inoculated was pricked through the centre with a sterile needle and the inoculum was then introduced on the point of another needle. The eye was marked with India ink. Control fruit were pricked through the eyes with a sterile needle but inoculum was not introduced.

After a week the fruit were harvested and sectioned, and isolations made from any lesions which had developed. The three organisms were readily recovered from the fruitlets which had been inoculated. The symptoms produced by the various fungi differed to some degree and are described below.

(1) *Fusarium* sp.—The lesions produced were brown in colour but very restricted and not extending far beyond the floral cavity. In some cases there was a slight brown discoloration of the fruit tissue but no distinct lesion.

(2) *Penicillium* sp.—The lesions produced varied somewhat in size but they were all of a rather dark brown colour and extended towards the core (Fig. 4).

(3) *Penicillium* sp.—The lesions varied from glossy-brown to reddish-brown in colour and they were quite extensive, spreading as far as the core.

In the control fruit pricked but uninoculated, only one fruitlet developed a lesion, and from this *Thielaviopsis paradoxa* was isolated.

In an endeavour to determine whether an injury to the lining of the floral cavity was needed before infection took place, further inoculations were carried out in the laboratory. Spore suspensions of the three organisms were each introduced with a micro-pipette into the floral cavities of 12 fruitlets. Where necessary the opening was enlarged with the point of a sterile needle. Sterile water was introduced into 12 fruitlets as a control. In each case six of the fruitlets were pierced with a sterile needle and six were not injured. The fruit were sectioned after seven days.

A lesion developed in 15 out of 18 fruitlets in which the floral cavity had been injured after inoculum was inserted, and in only one fruitlet that had not been damaged. The controls were unaffected.

Discussion.

Macroscopic examination of the floral cavities reveals the presence of fungal mycelium and spore masses growing on the floral remnants. *Penicillium* sp. can be readily detected.

Fig. 4.

Fruitlet Core Rot. Artificial infection with *Penicillium* sp. (Only the upper right fruitlet is in median section).

These organisms do not appear to be able to break down the fruit tissue unless the hard lining of the cavity is injured in some manner to allow their entry. Pricking with a sterile needle has been found sufficient. It is likely therefore that in the Brackenridge area the feeding activities of mealy bugs in the cavities provided the necessary site for infection of fruit.

Subsequent examination of affected fruit of the Smooth Cayenne variety from the Maroochy, Blackall Range, and Cooroy districts showed either mealy bugs or mites to be commonly present in the floral cavities above fruitlet core rot lesions. There is ample circumstantial evidence that mites are capable of playing a similar part to mealy bugs in providing a means of entry for the causal fungi into the fruit. Small growths such as those which develop on the surface of the fruit following certain climatic conditions may also provide the necessary injury to the lining of the floral cavities.

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THE FRUIT ROTS OF THE CUSTARD APPLE.

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SUMMARY.

Phomopsis anonaccarum Bondartzeva-Monteverde was isolated from fruit affected with black canker. Though pathogenicity tests were inconclusive, it is considered that the fungus is responsible for the disease. A marginal leaf scorch is attributed to the same organism.

The phycomycete Phytophthora palmivora Butler is responsible for purple blotch.

Botryodiplodia theobromae Pat. was shown by pathogenicity tests to be the cause of Diplodia rot.

INTRODUCTION.

The custard apple (*Annona squamosa* L.) is relatively free from serious diseases in southern Queensland. However, particularly under conditions of high rainfall, the fruit sometimes develop purplish black discoloured areas which either cause the fruit to fall off the tree or render them unfit for marketing. Lack of knowledge regarding the etiology of this trouble led to investigations being carried out over the past three seasons with a view to determining the agent or agents responsible.

During the course of these investigations three organisms have been found associated with somewhat different symptoms on custard apple fruit. These are *Phomopsis anonacearum* Bondartzeva-Monteverde, *Phytophthora palmivora* Butler, and *Botryodiplodia theobromae* Pat. Each of these diseases is now described.

BLACK CANKER.

This disease was recorded as early as 1926 and it has appeared spasmodically in each succeeding season. In the 1948–49 season, losses at an economic level were reported by many growers in the Sunnybank and Redland Bay areas near Brisbane. It has since been observed on fruit grown at Charters Towers in North Queensland.

Symptoms.

The condition is first recognised by the occurrence of purple lesions on the fruit, most commonly at or near the apical end. These vary greatly in size from small circular spots $\frac{1}{2}$-in. in diameter up to blotches covering half of the affected

fruit. The lesions become extremely hard and with age deep cracks develop in them (Fig. 1). Close examination reveals the presence of pycnidia which are slightly raised from the surface. Under moist conditions it is common to see white spore tendrils being extruded from the pycnidia (Fig. 2).

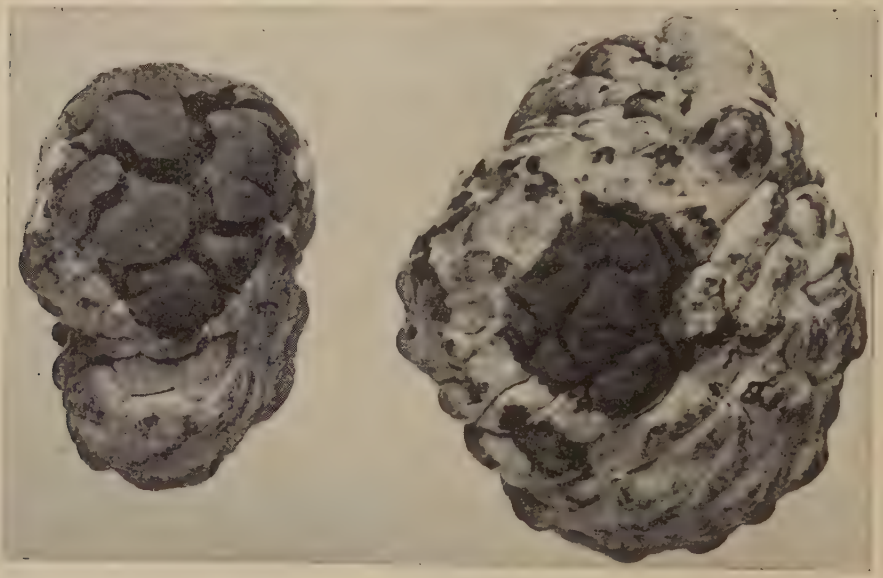

Fig. 1.
Typical Lesions of Black Canker.

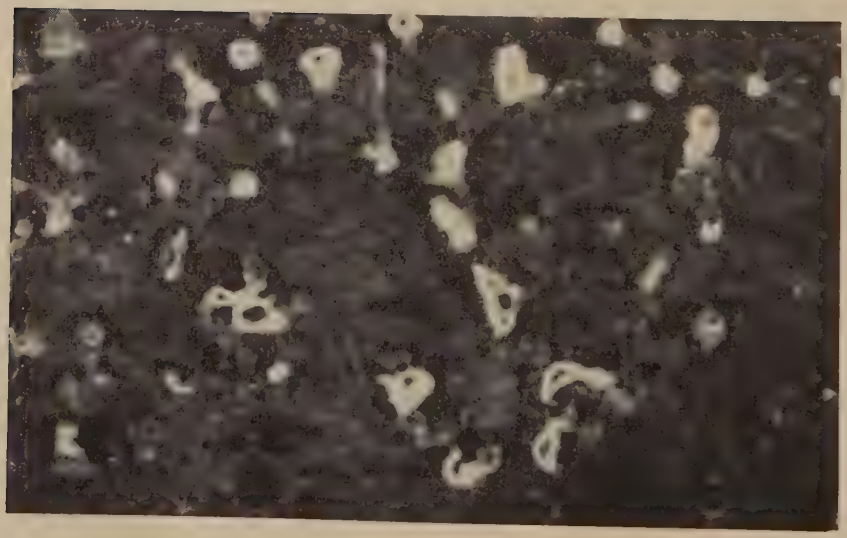

Fig. 2.
Microphotograph of Spore Tendrils of *Phomopsis anonacearum* on a Black Canker Lesion.

Internally the lesions are very characteristic, being brown in colour and usually no more than $\frac{1}{4}$-in. deep. The surrounding tissue is not affected and in mature fruit the remainder of the flesh is quite sound. However, the external discolouration renders affected fruit unfit for marketing.

Fruit at all stages of maturity and of all sizes have been found affected from as early as February through to the conclusion of the main harvesting season in June.

Isolations and Pathogenicity Tests.

A fungus was consistently isolated from the edge of the lesions and from pycnidia and spore tendrils taken from diseased material.

Over a period of three seasons numerous attempts have been made to infect fruit with this fungus.

A series of tests was conducted on immature fruit in the laboratory and in the field with cultures isolated from diseased specimens, employing the following technique :

The surface of the fruit was wiped over with alcohol and allowed to dry. Small areas were then marked off and from the centre of these a small cube about $\frac{1}{8}$-in. square was cut to a depth of $\frac{1}{4}$-in. and removed. Small pieces of inoculum from the cultures on potato-dextrose agar were then placed in the small cavity so created and the cube replaced. This was done in the field on fruit still on the trees, and in the laboratory on fruit held in a moist chamber.

A total of 36 such inoculations has been done into fruit at various stages of maturity, and on only two occasions has any degree of success been achieved.

One fruit was successfully infected in April, 1951. The tissue for about half an inch around the point of inoculation was discoloured purple and became hard. Close examination revealed pycnidia. This fruit was removed and placed in a moist chamber for 48 hours, during which time the lesion expanded somewhat and extended rather deeper into the tissue than naturally occurring lesions. The pycnidia were found to be producing typical pycnospores (Table 1).

Table 1.

SPORE MEASUREMENTS (in microns)—*Phomopsis* spp.

Source.	Pycnidia Diameter.	A Spores.	B Spores.
Black canker lesions (5)	96 to 150 x 60 to 105. Average 132 x 78 (50)	6·25 to 8·1 x 1·9 to 3. Average 6·6 x 2·4 (250)	
Black canker (artificial infection) (2)	96 to 150 x 60 to 120. Average 135 x 90 (10)	5 to 7·5 x 2·5 to 3·0. Average 6 x 2·75 (100)	16·5 to 24 x 1. Average 20·1 x 1 (50)
Marginal leaf scorch (2)	105 to 150 x 75 to 96. Average 117 x 81 (20)	5 to 7·5 x 1·9 to 3. Average 6·3 x 2·0 (100)	
Inoculated twigs (1)..	350 to 800. Average 585 (20)	5·25 to 7·5 x 2·25 to 3. Average 6·5 x 2·75 (25)	16·5 to 25·5 x 1. Average 19·5 x 1 (25)
Phomopsis anonae, on bark of *A. cherimoliae* (De Urries, 1951)	300 to 1,000	6·5 to 9·5 x 1·5 to 2·5	20 to 30 x 1 to 1·5. C spores. Average 13
Phomopsis anonacearum, on leaves of *A. cherimoliae* (Bondartzeva-Monteverde, 1936)	120 to 140	5 to 8 x 2 to 2·5 ..	

(Figures in brackets indicate the number of samples examined.)

A similar infection was obtained in one fruit during April, 1952, but with the rather more typical symptom of a hard shallow lesion.

A second technique employed involved placing portions of the cultures over small pin-hole injuries made with a needle and enclosing the fruit in a plastic bag to prevent rapid drying out of the culture. Negative results were obtained with this method.

During both seasons when these inoculations were conducted, natural infection was extremely low.

Following the failure to reproduce typical symptoms easily with culture inoculum, attempts were made to inoculate fruit with spore material. The techniques and the results were as follows :

1. Spore tendrils were picked off affected lesions and used as inoculum in much the same way as the cultures had been used previously. In addition, fruit in the field so inoculated were enclosed in moistened plastic bags to maintain humidity.

Negative results were obtained.

2. Spore suspensions were prepared in sterile water from fresh spore tendril material and used as inoculum in the field. The fruit were surface-sterilized with alcohol, sprayed with sterile water, then with spore suspension. and finally with sterile water again. A fine clinical atomiser was used for spraying. (Some of

the fruit were slightly injured with a needle before application of the spore suspension, because it was considered possible that infection occurred naturally through injuries.) After spraying, the fruit were enclosed in plastic bags which had been moistened internally to maintain humidity. Twenty-five fruit at all stages of maturity were treated in this way. A check was made on the suspension used in each case and spore germination was found to be very good after 24 hours.

Typical symptoms were not reproduced in any of the fruit so treated. However, five of the uninjured fruit developed an unusual " pin-head " spotted appearance which failed to develop further in the field. As these fruit approached maturity they were removed and placed in moist chambers in the laboratory. Under these conditions the fruit rapidly became discoloured (Fig. 3) and commenced to produce pycnidia from which spore tendrils were extruded. The spores were identical with those produced by the pycnidia on diseased fruit (Table 1). Control fruit, although finally discoloured purple, took much longer to reach this condition and failed to produce pycnidia.

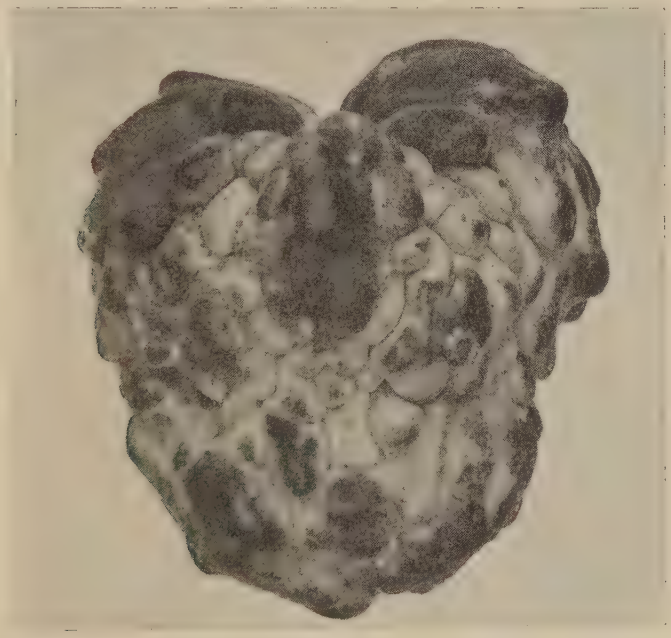

Fig. 3.

Discolouration Developing on Fruit Sprayed with a Suspension of *Phomopsis anonacearum* Spores.

3. Spore suspensions prepared as for (2) were applied to fruit held in moist chambers in the laboratory.

Similar results were obtained, the inoculated fruit turning purple very quickly and producing pycnidia and typical spores. One such fruit produced pycnidia bearing both A spores and B spores (See later).

Marginal Leaf Spotting and its Relationship to Black Canker of the Fruit.

An interesting recording of a marginal leaf scorch was made on custard apple leaves in 1952. A large number of leaves on many trees were exhibiting dead areas, particularly at the tips of the leaves (Fig. 4). Close examination of these demonstrated the presence of pycnidia, which proved to contain A spores similar in shape and size to those previously recorded on black canker lesions. Again under moist conditions the spores were extruded from the pycnidia in white tendrils. Further investigations demonstrated that this leaf condition was widespread, particularly as the season advanced.

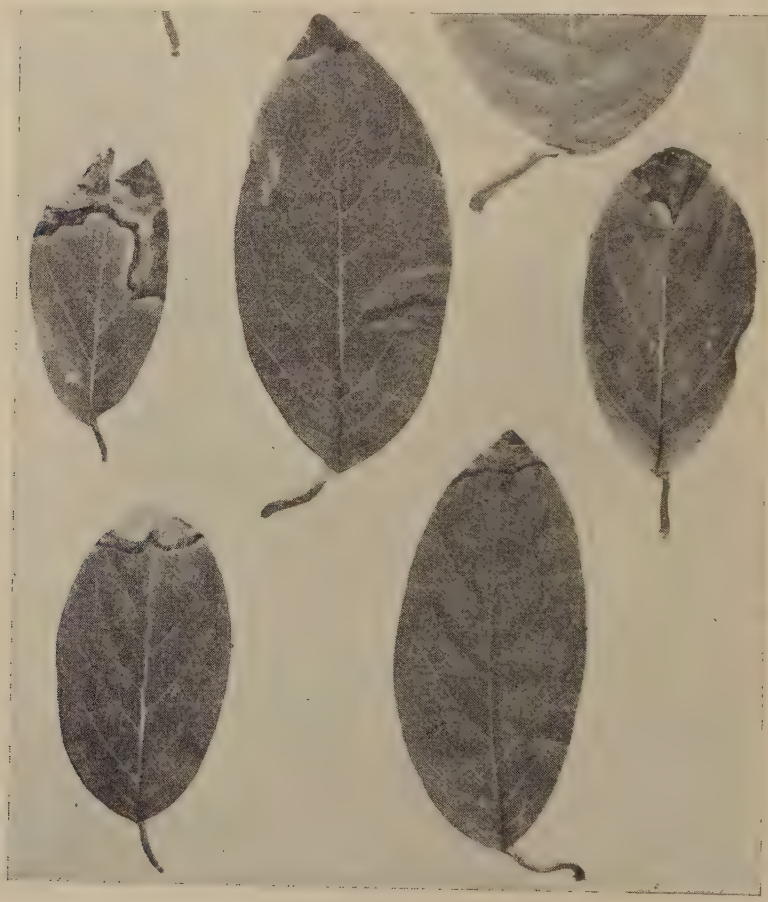

Fig. 4.

Marginal Leaf Scorch with Which *Phomopsis anonacearum* is Associated.

A thorough examination of such material and of dead leaf tissue from beneath the tree failed to reveal any fructification which might prove to be the perfect stage of the fungus.

Healthy and affected leaves were then submitted to various laboratory treatments similar to those used by Kiely (1948) to induce the production of *Guignardia citricarpa* on citrus leaves. Abundant pycnidia were produced by this means on all diseased and some apparently healthy leaves, but no perfect stage was encountered in any of the treatments.

Stem material was examined for possible fructifications of the fungus but none was found.

Pathogenicity tests were then conducted with the diseased leaf material. A spore suspension was made from spore tendrils and fruit inoculated by the spray technique already described. No infection was obtained.

In a further attempt, infected leaves which had been surface-sterilized were tied so as to be in close contact with healthy surface-sterilized fruit. The whole fruit was then wrapped in moist cotton-wool and enclosed in a plastic cover to maintain humidity. A similar technique had been employed by Kiely to

Fig. 5.
Lesions Developing on Custard Apple Fruit Inoculated with Diseased Leaf Material.

test the pathogenicity of *G. citricarpa*. After seven days definite symptoms were recorded on three of the eight fruit so inoculated. Definite hard purple lesions producing pycnidia appeared (Fig. 5) and the fungus was isolated from them.

Attempts were made to infect healthy leaves on the trees with the fungus obtained from black canker lesions and marginal leaf spot. Spore suspensions were prepared and fresh young leaves sprayed and enclosed with plastic bags as was done in the fruit inoculations. Negative results were obtained.

One series of leaves showing marginal scorch was retained in a dried condition in the laboratory for 12 months. At the end of this period these leaves were moistened and incubated at 27·5°C. The pycnidia present produced spores abundantly.

Although the results of the artificial infection experiments were disappointing, the universal association of this fungus producing pycnidia with the disease in the field, coupled with the knowledge that species of this type are often difficult to deal with under artificial conditions, leaves little doubt that this organism is responsible for black canker.

There is also little doubt that the organism associated with the marginal leaf scorch is identical with the black canker fungus.

Description and Identification of the Fungus.

The organism on potato-dextrose agar produces relatively slow-growing white colonies with a floury surface. Concentric rings are built up in these colonies and with age the substrate becomes a dark brown. After approximately 90 days pycnidia develop on the colonies, but no pycnospores have been found in them.

The fungus has a temperature range from a minimum of 7°C. to a maximum of 35°C., with the optimum level of growth occurring at 28·5°C.

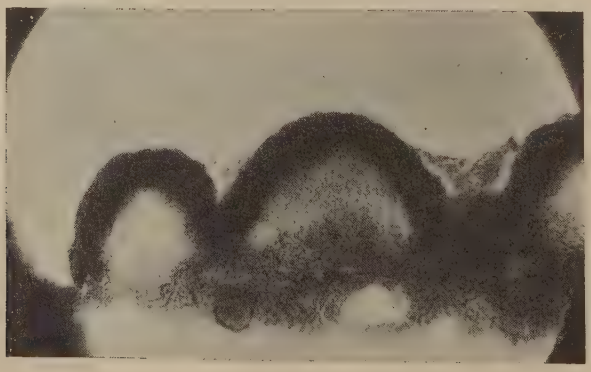

Fig. 6.

Microphotograph of Pycnidia of *Phomopsis anonacearum* on Diseased Fruit.

On black canker lesions the fungus produces pycnidia which are erumpent (Fig. 6), usually solitary and commonly ostiolate. The walls are carbonaceous black by transmitted light. These pycnidia produce spores which are rather variable in shape but are mostly fusiform, elliptical and commonly binucleate. Sporophores are simple, rod shaped about 10 μ in length and slightly narrower than the spores.

Specimens were submitted to the Commonwealth Mycological Institute, Kew, which reported that pycnidia in culture were producing both the A spores described above and B spores. The latter were described as curved to hamate, measuring 20–30 μ long and less than 1 μ broad. Such spores have never been encountered on naturally infected fruit in Queensland but have recently been found on artificially inoculated fruit. Measurements of pycnidia and both types of spores appear in Table 1.

On marginal leaf scorch lesions the pycnidia are normally immersed (Fig. 7), have a black carbonaceous wall and are ostiolate. Measurements of pycnidia and A spores appear in Table 1. No B spores have been found in these pycnidia.

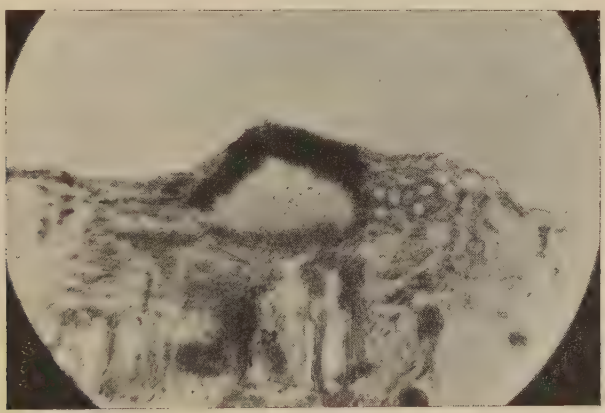

Fig. 7.

Microphotograph of Pycnidia of *Phomopsis anonacearum* on Leaf Lesion.

The fungus has been identified as *Phomopsis anonacearum* Bondartzeva-Monteverde by M. B. Ellis and E. W. Mason of the Commonwealth Mycological Institute. This species was described from hothouses in Leningrad as causing a leaf disease similar to that described here as marginal leaf scorch (Bondartzeva-Monteverde, Gutner and Novosselova 1936), but no previous record of it causing a fruit disease can be found. In the original description no mention is made of B spores and evidence in Queensland is that their occurrence under natural conditions, if ever, is rare.

Phomopsis anonae De Urries has been described on the bark of *Annona cherimoliae* in Valencia (De Urries, 1951). To compare *P. anonacearum* with the description given by De Urries, custard apple twigs were placed in glass tubes

so that their bottoms were in sand. After autoclaving, sterile water was added to the sand, and the stems inoculated with cultures of *P. anonacearum* isolated from black canker lesions. After 26 days a number of twigs were covered with a white weft of mycelium, and large stromatic structures which proved to be pycnidia were evident on the bark.

These pycnidia were usually erumpent and extremely variable in shape and size from compressed globular to flask-shaped. Often there was a distinct neck, in some cases more than one, opening with a round ostiole. Often, white spore tendrils were seen extruding from these pycnidia. The walls of the fructifications were an olivaceous brown and much thickened, particularly towards the top (Fig. 8). The bottom wall was often elevated in sections, giving a divided appearance to the pycnidial cavity. Spores of two types, A and B, similar to those previously described were produced.

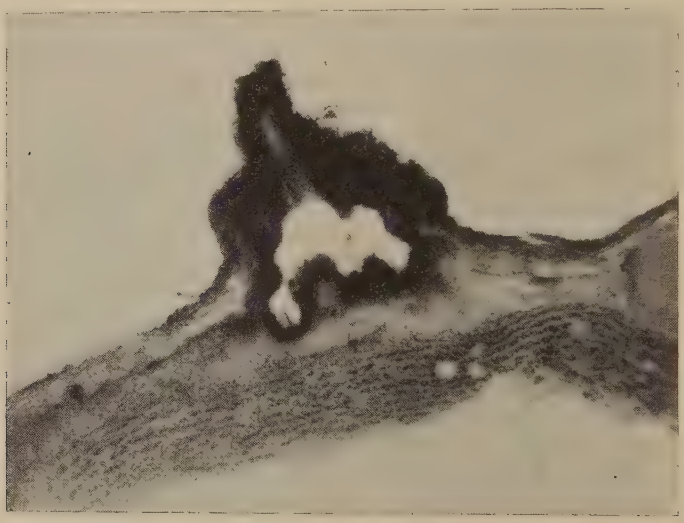

Fig. 8.

Microphotograph of Pycnidia of *Phomopsis anonacearum* on Inoculated Stems.

These fructifications differ somewhat from the normal pycnidia found on black canker lesions and appear identical with those described by De Urries for *P. anonae*. De Urries describes a third spore type which was not found here and his measurements for A spores are slightly larger (Table 1). However, it does seem very probable that *Phomopsis anonae* De Urries is a synonym of *Phomopsis anonacearum* Bondartzeva-Monteverde.

A species of *Phoma* has been recorded causing a leaf spot on custard apple in New South Wales (Noble, Hynes, McCleery and Birmingham, 1934).

A fungus identical with *Phomopsis anonacearum* has recently been isolated from diseased fruit of bullock's heart (*Annona reticulata* L.) growing in North Queensland.

Field Infection.

The evidence indicates that the occurrence of *P. anonacearum* on leaf tissue provides a very effective means of overwintering of the fungus. The difficulty of obtaining infection artificially with pycnospore suspensions throws doubt on the importance of this stage in primary infection. However, field evidence shows that the disease is most severe under conditions of high rainfall when the humidity is high and when pycnidia would be expected to be liberating many spores. The importance of a possible perfect stage in infection has not been overlooked but no evidence for the occurrence of such a stage has been obtained.

There is a possibility that mummified fruit lying under the trees may be a source of infection, but although many of these have been examined, none of the *P. anonacearum* or related fructifications have been encountered. Those examined have invariably been overgrown with *Botryodiplodia theobromae* and saprophytic organisms.

PURPLE BLOTCH.

This disease was first observed at Sunnybank in February 1950, when a grower reported considerable fruit fall. Since then affected fruit have been found on many farms. Because of its ability to attack very young fruit, the disease causes losses of which the grower is often quite unaware. Severe outbreaks have been very restricted but it remains a potential danger during periods of high rainfall.

Symptoms.

The presence of the disease in a plantation is first indicated by excessive fall of small immature fruit. Close examination of the trees reveals that many of the fruit show purple lesions which commence as small spots but rapidly expand until most of the fruit surface may be discoloured. The affected portion of the fruit does not become hard and the lesion has a somewhat indistinct margin (Fig. 9).

Infected fruit falls at a touch and a brown discolouration is seen extending from the fruit into the fruit stalk. During a severe outbreak many discoloured fruit can be seen on the ground. These quickly become " mummified " and are difficult to distinguish from fruit that fell during the previous season.

Fruit of any size may be infected, with specimens as small as 1-in. in diameter being commonly found. The disease develops rapidly, careful tagging at Sunnybank showing that falling occurs within four days of the first appearance of purple discolouration on small fruit. Where clean cultivation is not practised it is easy to visualize severe losses occurring before the grower realises the presence of the disease.

Affected fruit are usually completely discoloured brown internally even prior to fall (Fig. 10). This is in direct contrast to the shallow lesions characteristic of black canker.

Fig. 9.
Purple Blotch—Field Symptom.

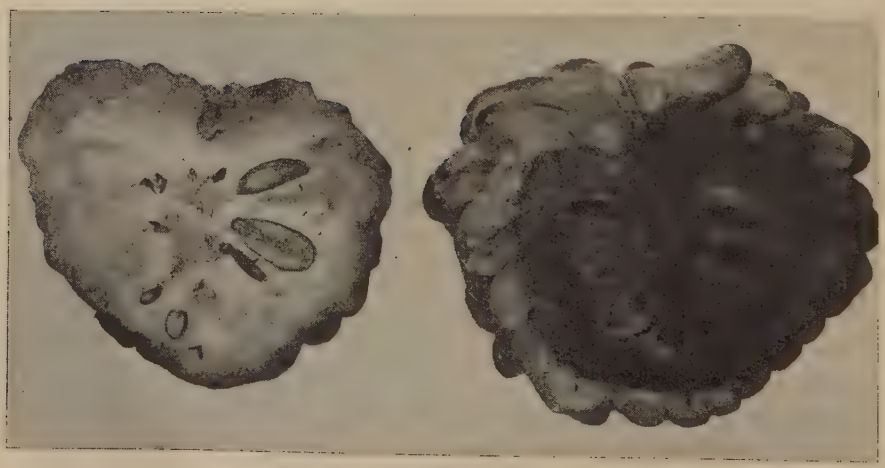

Fig. 10.
Internal Discolouration Produced on the Right by *Phytophthora palmivora* and on the Left by *Phomopsis anonacearum* (lesion on lower left hand side).

The disease has been recorded in the field during February and March and it has been possible to obtain positive artificial infection as late as May.

Isolations and Pathogenicity Tests.

A white phycomycete has been consistently isolated from the brown discoloured tissue of diseased fruit.

The technique envolving the removal of cubes, as described earlier, was used for testing the pathogenicity of these isolates.

Fruit ranging in size from as small as 1-in. in diameter were inoculated in the field and positive infection was obtained from February until May. The small fruit became discoloured purple and fell 4–5 days after inoculation. Larger fruit took 10 days to fall in February and up to 20 days later in the season. Typical external and internal symptoms were reproduced (Fig. 11).

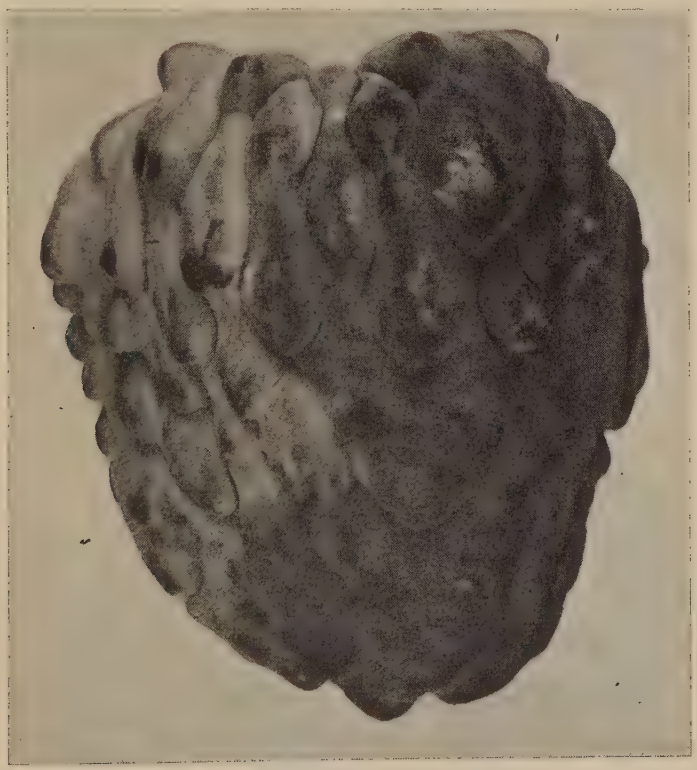

Fig. 11.

Symptoms Following Inoculation with *Phytophthora palmivora.*

In the laboratory, inoculated fruit rapidly developed large lesions and became discoloured internally.

Identity and Description of the Fungus.

A culture of the phycomycete forwarded to the Commonwealth Mycological Institute, Kew, was identified by Miss G. M. Waterhouse as *Phytophthora palmivora* Butler and classified in the "Cacao" group. The fungus has a temperature range from a minimum of 10°C. to a maximum of 35°C., with an optimum at approximately 31°C. on malt extract agar. No sexual bodies are produced on solid oatmeal agar media, but there is a ready formation of clamydospores and ovoid to elliptical papillate sporangia. Only chlamydospores and sporangia are formed when sterile mycelium is transferred from pea broth to distilled water. On potato-dextrose agar the colonies are relatively slow growing, flat, with a slight development of white aerial mycelium.

P. palmivora (syn. *P. parasitica* Dastur) has been previously recorded in Queensland on citrus and recently on rhubarb and rosella by G. D. Bowen (Queensland Department of Agriculture and Stock, unpublished records, 1952).

Phytophthora sp. has been recorded in New South Wales in association with a leaf spot and scald, twig dieback and brown rot in custard apple (New South Wales Department of Agriculture, 1951).

Field Infection.

The disease is restricted to the fruit on the lower branches of the tree and has been recorded only after heavy falls of rain. This evidence would suggest that the fungus is a soil inhabitant and gains access to the lower fruit during periods of rainy weather through rain splash. The only severe outbreak of the disease has occurred on a property where poultry running in the orchard maintain a bare surface on the soil. Under such conditions the splashing of infective material on to fruit would be greatly assisted.

DIPLODIA ROT.

This disease is frequently encountered in custard apple plantations but is not the cause of much economic loss. It has been recorded as far north as Charters Towers and is prevalent in the main areas surrounding Brisbane, particularly in rather neglected plantations.

Symptoms.

The most usual condition produced by this disease is a mummification of fruit which remain attached to the tree. The disease usually commences as a small lesion which rapidly expands and becomes hard and cracked, resembling somewhat the external symptoms produced by black canker. Such lesions usually cover at least half of the fruit and numerous large pycnidia are often visible on their surface (Fig. 12).

Fig. 12.

Fruit Rot Caused by *Botrydiplodia theobromae.*

The lesions extend deep into the internal tissue of the fruit, often completely discolouring it and producing a brown corky condition similar to that associated with purple blotch (Fig. 13).

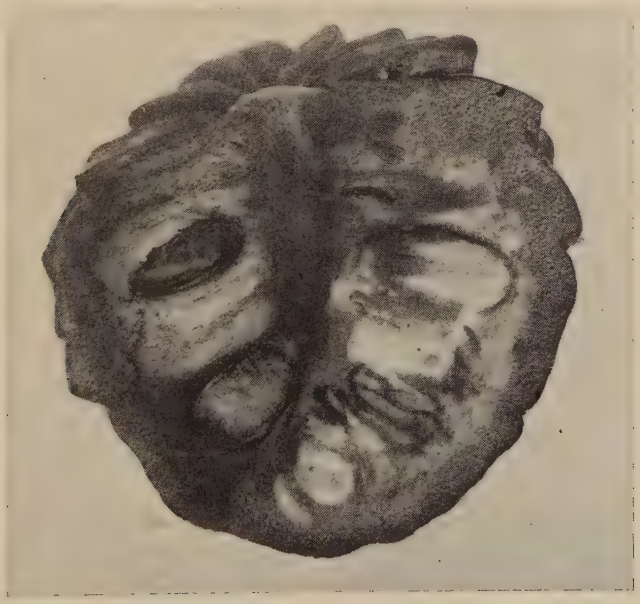

Fig. 13.

Internal Discolouration Produced by *Botryodiplodia theobromae.*

Isolations and Pathogenicity Tests.

A fungus was first isolated from the discoloured tissue of affected fruit by G. D. Bowen in 1950 (Queensland Department of Agriculture and Stock, unpublished records, 1950). Single spore isolates and pycnidial isolates have since yielded the same organism.

The technique of placing culture inoculum in small injuries, as described earlier, was used to test the pathogenicity of several isolates. Fruit in the field and in the laboratory were successfully inoculated by this method. In the laboratory, lesions were large, spreading rapidly over the fruit and quickly discolouring all the tissue. Numerous pycnidia exuding tendrils were produced. The fungus was re-isolated. In the field, the inoculations in March and April were successful, while later inoculations under colder weather conditions were a failure. The infected fruit first produced a small purple lesion, which, after a slow initial period of 14 days, rapidly spread over the whole of the fruit. Unlike purple blotch, the fruit remained attached to the tree. Pycnidia were also produced on these fruit. The fungus was re-isolated.

Description and Identification of the Fungus.

On potato-dextrose agar the fungus produces quick-growing colonies with at first rather sparse, white to grey aerial mycelium, which becomes woolly and black with age. The substrate quickly changes from light green to a deep greenish black colour.

The fungus has a temperature range from a minimum of 9·5°C. to a maximum of 40°C. Optimum development is obtained at a temperature of approximately 29°C.

On potato-dextrose agar the fungus fails to produce any fruiting bodies when grown in darkness. Under laboratory light at room temperatures, stromatic structures are produced after three weeks. These structures are up to 3 mm. in height and have numerous pycnidia embedded in them.

The pycnidia occurring on infected fruit on the tree are usually seperate, globose in shape and occur superficially on the surface. They produce elliptical spores which are at first hyaline and non-septate but become dark brown and one-septate with age. Distinct longitudinal striations are visible on the exospore.

On some mummified fruit found on the ground the stromatic structures described above as occurring on culture media have been observed. It has been possible to produce these on infected fruit by placing them in a moist chamber in the laboratory. Spores similar to those occurring in the individual pycnidia are produced from these stromatic structures (Fig. 14). Table 2 records the relative sizes of the spores and pycnidia obtained.

Fig. 14.
Microphotograph of a Section Through a Stroma of *Botryodiplodia theobromae.*

Table 2.

SPORE MEASUREMENTS (in microns)—*Botryodiplodia theobromae.*

Source.	Pycnidia Diameter.	Hyaline Non Septate Spores.	One Septate Dark Spores.
Diseased fruit (single pycnidia) (5)	220 to 450. Average 240 (40)	21 to 30 x 12 to 16·5. Average 25 x 14·5 (125)	21 to 30 x 12 to 16·5. Average 25 x 14·5 (125)
Diseased fruit (Stroma) (2)		Average 25 x 14·5 (25)	Average 25 x 14·5 (25)
Culture (Stroma) (2)		19·5 to 27 x 12 to 15. Average 24·4 x 12·2 (50)	21 to 28·5 x 12 to 15. Average 24·9 x 13·5 (50)
Stem dieback (2) ..	225 to 450. Average 275 (10)	Average 24 x 12·5 (50)	Average 24 x 12 (50

(Figures in brackets indicate the number of samples examined.)

The spore measurements and presence of stromatic structures place the fungus as *Botryodiplodia theobromae* Pat. This has been recorded previously under the synonym *Lasiodiplodia theobromae* on fruit of *Annona squamosa* in the Philippine Islands (Philippine Islands Bureau of Agriculture, 1926) and as a wound parasite and saprophyte on many tropical crops.

Field Infection.

The fruiting bodies of this fungus, both in the single pycnidial form and in the large stromatic structures, have been found frequently associated with dieback in custard apple trees (Table 2). It has not been determined whether this organism is acting as a parasite or merely as a secondary invader of the dying stem tissue.

The widespread occurrence of the organism on dead wood and in mummified fruit assures an abundant supply of infective material. The disease on the fruit is never in serious proportions and it appears likely that the organism is acting here merely as a wound parasite.

Diplodia natalensis has been recorded causing stem blight of *A. squamosa* in Texas (Freeman Weiss, 1950). As both the simple pycnidia characteristic of *D. natalensis* and the stromatic structure characteristic of *B. theobromae* have been found on local diseased stem material, it is probable that the disease reported in Texas is a similar condition.

In Queensland, *Botryodiplodia theobromae* has recently been recorded on the fruit of bullock's heart (*Annona reticulata*) in the Barron River area. It has previously been recorded on *Araucaria cunninghamii* (Young, 1936), while *D. natalensis* has been obtained from *Pinus* sp. (Young, 1936) and is common on citrus fruit, causing a stem end rot.

Lemons inoculated with the fungus isolated from custard apples produced stem end lesions similar to those caused by *D. natalensis*.

GENERAL REMARKS AND CONTROL.

The total area planted to custard apples in Queensland approximates 400 acres, made up in the main of small areas from 1 acre to 10 acres in size. The main producing localities are in the Brisbane district, where this fruit is commonly grown in association with such crops as vegetables and papaws.

In the past none of these diseases on custard apples has been severe enough to warrant expensive control measures being undertaken. Black canker does at times cause losses at an economic level. Purple blotch, although capable of producing severe losses, has been restricted to a small number of farms. However, there are difficulties encountered in spraying custard apple trees on account of their large size and the fact that the fruit are hidden in the rather profuse foliage. For the present, attention to cultural methods, particularly orchard hygiene involving removal of dead wood and mummified fruit, should assist in minimising the incidence of these diseases.

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